Amendments to the Claims:

This listing of claims will replace all prior versions, and listings, of claims in the application:

Claims 1-39 (canceled)

Add the following claims 40-51.

Claims

40. A method of identifying subjects having a high or low drug metabolizing phenotype associated with cytochrome CYP3A5 expression from variant or wild-type DNA sequences, which method comprises the steps of:

obtaining a suitable sample from the subject; and

screening the genomic DNA from said sample for the presence or absence of both the variants $T_{-475}G$ and $A_{-147}G$ in the transcriptional regulatory region of CYP3A5.

41. A method according to claim 40 wherein during the screening process, the genomic DNA is amplified using oligonucleotide molecules which are capable of hybridizing selectively to the wild-type or variant sequences respectively such that generation of amplified DNA from said respective molecules will indicate whether said wild-type or said variant is present.

42. A method of identifying the polymorphic variants $T_{-475}G$ and $A_{-147}G$ of the transcription regulatory region of a sample of either variant or wild-type DNA encoding cytochrome CYP3A5 said method comprising the steps of:

1) subjecting the sample DNA to amplification using oligonucleotide molecules which are capable of selectively hybridizing to the wild-type and/or said one or more variant sequences, which molecules are such that they can introduce a restriction site in one of said amplified wild type or variant sequences, and

2) subjecting amplified DNA from step 1 to restriction with an enzyme which cleaves at said restriction site to provide a restriction digest indicative of the presence or absence of said mutation.

43. A method according to claim 42 wherein the oligonucleotide molecule introduces a restriction site in a region corresponding to a recognition site for a transcription factor of said regulatory region.

44. A method according to claim 43 wherein the oligonucleotide molecule introduces a restriction site in a region corresponding to an activator protein-3 motif (AP-3) and/or a basic transcription element (BTE).

45. A method according to claim 44 wherein the oligonucleotide molecule is capable of introducing a restriction site only when the wild type A nucleotide is present at position -147 of the transcription regulatory region.

46. A method according to claim 45 wherein the restriction site is for the Tai I restriction enzyme.

47. A method according to claim 46 wherein said oligonucleotide molecule comprises the sequence designated 3A5R1 illustrated in Figure 6.

48. A method according to claim 44 wherein the oligonucleotide molecule is capable of introducing a restriction site when the wild type T nucleotide is present at position -475 of the regulatory control region.

49. A method according to claim 48 wherein the restriction site is for the Alu I enzyme.

50. A method according to claim 49 wherein the oligonucleotide molecule comprises the sequence designated 3A5F2 illustrated in Figure 6.

51. A method of diagnosing susceptibility of an individual to a disease associated with environmental toxins or pro-carcinogens metabolized by CYP3A5, comprising

1) identifying subjects having a high or low drug metabolizing phenotype associated with cytochrome CYP3A5 expression from variant or wild-type DNA sequences,

2) screening a suitable sample of the genomic DNA from of those subjects for the presence or absence of both variants $T_{-475}G$ and $A_{-147}G$ of the transcription regulatory region of the sequence encoding CYP3A5 the sequence of which region is illustrated in Figure 7.